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DATE: Friday, May 06, 2005 [Printable Copy](#) [Create Case](#)

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DB=USPT; PLUR=YES; OP=OR

<u>L8</u>	L7 and wrana.in.	0	<u>L8</u>
<u>L7</u>	L6 and l5	51	<u>L7</u>
<u>L6</u>	thomsen.in.	676	<u>L6</u>
<u>L5</u>	screening method and L4	394742	<u>L5</u>
<u>L4</u>	L3 and Smurf activity	390287	<u>L4</u>
<u>L3</u>	l1 and L2	43178	<u>L3</u>
<u>L2</u>	PPXY domain and Smad polypeptide	49936	<u>L2</u>
<u>L1</u>	smurf activity	390300	<u>L1</u>

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Search Results - Record(s) 1 through 10 of 51 returned.

☐ 1. Document ID: US 6846919 B2

L7: Entry 1 of 51

File: USPT

Jan 25, 2005

US-PAT-NO: 6846919

DOCUMENT-IDENTIFIER: US 6846919 B2

TITLE: Non-endogenous, constitutively activated human serotonin receptors and small molecule modulators thereof

DATE-ISSUED: January 25, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Behan; Dominic P	San Diego	CA		
Chalmers; Derek T	Solana Beach	CA		
Liaw; Chen W	San Diego	CA		
Russo; Joseph F	San Diego	CA		
Thomsen; William J	Del Mar	CA		

US-CL-CURRENT: 536/23.1; 435/235.1, 435/320.1, 435/325, 435/69.1, 435/7.1, 530/300, 530/350, 536/23.5

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	WMC	Draw Desc	Ima
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☐ 2. Document ID: US 6745333 B1

L7: Entry 2 of 51

File: USPT

Jun 1, 2004

US-PAT-NO: 6745333

DOCUMENT-IDENTIFIER: US 6745333 B1

TITLE: Method for detecting unauthorized network access by having a NIC monitor for packets purporting to be from itself

DATE-ISSUED: June 1, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thomsen; Brant D.	Sandy	UT		

US-CL-CURRENT: 713/201; 713/160, 713/161, 713/168, 713/200

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	WMC	Draw Desc	Ima
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☐ 3. Document ID: US 6719968 B2

L7: Entry 3 of 51

File: USPT

Apr 13, 2004

US-PAT-NO: 6719968
DOCUMENT-IDENTIFIER: US 6719968 B2

TITLE: Tendon-inducing compositions

DATE-ISSUED: April 13, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Celeste; Anthony J.	Hudson	MA		
Wozney; John M.	Hudson	MA		
Rosen; Vicki A.	Brookline	MA		
Wolfman; Neil M.	Dover	MA		
<u>Thomsen</u> ; Gerald H.	Port Jefferson	NY		
Melton; Douglas A.	Lexington	MA		

US-CL-CURRENT: 424/85.1; 514/12, 514/2, 530/350, 530/351, 530/395, 530/397, 530/399

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KMC	Draw Desc	Ima
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☐ 4. Document ID: US 6689170 B1

L7: Entry 4 of 51

File: USPT

Feb 10, 2004

US-PAT-NO: 6689170
DOCUMENT-IDENTIFIER: US 6689170 B1

TITLE: Implant element

DATE-ISSUED: February 10, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Larsson; Cecilia	S-412 74 Goteborg			SE
<u>Thomsen</u> ; Peter	S-421 67, Goteborg			SE

US-CL-CURRENT: 623/23.53; 623/16.11

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KMC	Draw Desc	Ima
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☐ 5. Document ID: US 6682892 B2

L7: Entry 5 of 51

File: USPT

Jan 27, 2004

US-PAT-NO: 6682892
DOCUMENT-IDENTIFIER: US 6682892 B2

TITLE: Method for treating herpes viruses

DATE-ISSUED: January 27, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Homa; Fred L.	Kalamazoo	MI		
Wathen; Michael W.	Portage	MI		
Hopkins; Todd A.	Galesburg	MI		

US-CL-CURRENT: 435/6; 435/235.1, 435/325, 435/5

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KMC	Draw Desc	Ima
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☐ 6. Document ID: US 6682649 B1

L7: Entry 6 of 51

File: USPT

Jan 27, 2004

US-PAT-NO: 6682649

DOCUMENT-IDENTIFIER: US 6682649 B1

TITLE: Substrate and a method for determining and/or monitoring electrophysiological properties of ion channels

DATE-ISSUED: January 27, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Petersen; Jon Wulff	Lyngby			DK
Telleman; Pieter	Lyngby			DK
Hansen; Ole	Lyngby			DK
Christophersen; Palle	Ballerup			DK
Bech; Morten	Ballerup			DK
Olesen; Soren Peter	Ballerup			DK
Due; Jorgen	Ballerup			DK
Thomsen; Lars	Ballerup			DK

US-CL-CURRENT: 205/777.5; 204/403.01, 422/82.01

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KMC	Draw Desc	Ima
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☐ 7. Document ID: US 6653886 B1

L7: Entry 7 of 51

File: USPT

Nov 25, 2003

US-PAT-NO: 6653886

DOCUMENT-IDENTIFIER: US 6653886 B1

TITLE: Power saving amplifier with selectable current levels

DATE-ISSUED: November 25, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Lee; Wai Laing	Austin	TX		
Kasha; Dan	Providence	RI		
Thomsen; Axel	Austin	TX		

US-CL-CURRENT: 327/374; 327/170, 327/337, 330/9

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KMC	Draw Desc	Ima
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☐ 8. Document ID: US 6639411 B1

L7: Entry 8 of 51

File: USPT

Oct 28, 2003

US-PAT-NO: 6639411

DOCUMENT-IDENTIFIER: US 6639411 B1

TITLE: Microactuated suspension motor failure detection system

DATE-ISSUED: October 28, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
<u>Thomsen</u> ; Jeffrey E.	Cosmos	MN		

US-CL-CURRENT: 324/537; 324/772

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KMC	Draw Desc	Ima
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☐ 9. Document ID: US 6614285 B2

L7: Entry 9 of 51

File: USPT

Sep 2, 2003

US-PAT-NO: 6614285

DOCUMENT-IDENTIFIER: US 6614285 B2

TITLE: Switched capacitor integrator having very low power and low distortion and noise

DATE-ISSUED: September 2, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Lee; Wai Laing	Austin	TX		
Kasha; Dan	Austin	TX		
<u>Thomsen</u> ; Axel	Austin	TX		

US-CL-CURRENT: 327/337; 327/374, 330/9, 341/172

Full	Title	Citation	Front	Review	Classification	Date	Reference			Claims	KMC	Draw Desc	Ima
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☐ 10. Document ID: US 6555350 B2

L7: Entry 10 of 51

File: USPT

Apr 29, 2003

US-PAT-NO: 6555350

DOCUMENT-IDENTIFIER: US 6555350 B2

TITLE: Method for processing lignocellulosic material

DATE-ISSUED: April 29, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Ahring; Birgitte Ki.ae buttet.d.r	DK-2970 H.o slashed.rsholm			DK
<u>Thomsen</u> ; Anne Belinda	Roskilde			DK

US-CL-CURRENT: 435/162; 435/132, 435/161

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Terms	Documents
L6 and L5	51

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=> s PPXY domain
L1 4 PPXY DOMAIN

=> d l1 ti abs ibib tot

L1 ANSWER 1 OF 4 MEDLINE on STN
TI Enac degradation in A6 cells by the ubiquitin-proteosome proteolytic pathway.
AB Amiloride-sensitive epithelial Na(+) channels (ENaC) are responsible for trans-epithelial Na(+) transport in the kidney, lung, and colon. The channel consists of three subunits (alpha, beta, gamma) each containing a proline rich region (PPXY) in their carboxyl-terminal end. Mutations in this **PPXY domain** cause Liddle's syndrome, an autosomal dominant, salt-sensitive hypertension, by preventing the channel's interactions with the ubiquitin ligase Neural precursor cell-expressed developmentally down-regulated protein (Nedd4). It is postulated that this results in defective endocytosis and lysosomal degradation of ENaC leading to an increase in ENaC activity. To show the pathway that degrades ENaC in epithelial cells that express functioning ENaC channels, we used inhibitors of the proteosome and measured sodium channel activity. We found that the inhibitor, MG-132, increases amiloride-sensitive trans-epithelial current in Xenopus distal nephron A6 cells. There also is an increase of total cellular as well as membrane-associated ENaC subunit molecules by Western blotting. MG-132-treated cells also have increased channel density in patch clamp experiments. Inhibitors of lysosomal function did not reproduce these findings. Our results suggest that in native renal cells the proteosomal pathway is an important regulator of ENaC function.

ACCESSION NUMBER: 2001308612 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11278712
TITLE: Enac degradation in A6 cells by the ubiquitin-proteosome proteolytic pathway.
AUTHOR: Malik B; Schlanger L; Al-Khalili O; Bao H F; Yue G; Price S R; Mitch W E; Eaton D C
CORPORATE SOURCE: Department of Physiology and Renal Division, Emory University, Atlanta, Georgia 30322, USA..
bmalik@ccms-renal.physio.emory.edu
CONTRACT NUMBER: DK 37963-14 (NIDDK)
DK-37175 (NIDDK)
DK-50268-4 (NIDDK)
DK-50740 (NIDDK)
SOURCE: Journal of biological chemistry, (2001 Apr 20) 276 (16) 12903-10. Electronic Publication: 2001-01-26.

Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010604
Last Updated on STN: 20030105
Entered Medline: 20010531

L1 ANSWER 2 OF 4 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

TI ENaC Degradation in A6 Cells by the Ubiquitin-Proteasome Proteolytic Pathway.

AB Amiloride-sensitive epithelial Na(+) channels (ENaC) are responsible for trans-epithelial Na(+) transport in the kidney, lung, and colon. The channel consists of three subunits (α , β , γ) each containing a proline rich region (PPXY) in their carboxyl-terminal end. Mutations in this **PPXY domain** cause Liddle's syndrome, an autosomal dominant, salt-sensitive hypertension, by preventing the channel's interactions with the ubiquitin ligase Neural precursor cell-expressed developmentally down-regulated protein (Nedd4). It is postulated that this results in defective endocytosis and lysosomal degradation of ENaC leading to an increase in ENaC activity. To show the pathway that degrades ENaC in epithelial cells that express functioning ENaC channels, we used inhibitors of the proteasome and measured sodium channel activity. We found that the inhibitor, MG-132, increases amiloride-sensitive trans-epithelial current in Xenopus distal nephron A6 cells. There also is an increase of total cellular as well as membrane-associated ENaC subunit molecules by Western blotting. MG-132-treated cells also have increased channel density in patch clamp experiments. Inhibitors of lysosomal function did not reproduce these findings. Our results suggest that in native renal cells the proteasomal pathway is an important regulator of ENaC function.

ACCESSION NUMBER: 2003459568 EMBASE
TITLE: ENaC Degradation in A6 Cells by the Ubiquitin-Proteasome Proteolytic Pathway.
AUTHOR: Malik B.; Schlanger L.; Al-Khalili O.; Bao H.-F.; Yue G.; Price S.R.; Mitch W.E.; Eaton D.C.
CORPORATE SOURCE: B. Malik, Dept. of Physiology, Ctr. for Cell and Molec. Signaling, Physiology Bldg., 1648 Pierce Dr., Atlanta, GA 30322, United States. bmalik@ccms-renal.physio.emory.edu
SOURCE: Journal of Biological Chemistry, (20 Apr 2001) Vol. 276, No. 16, pp. 12903-12910.
Refs: 28
ISSN: 0021-9258 CODEN: JBCHA3

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
022 Human Genetics
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20031204
Last Updated on STN: 20031204

L1 ANSWER 3 OF 4 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI Novel isolated Smurf protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.

AN 2001-071267 [08] WPIDS

AB WO 200077168 A UPAB: 20011129

NOVELTY - An isolated Smurf1 or Smurf2 protein (I), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) an isolated nucleic acid (II) encoding (I);
- (2) a vector (III) comprising (II);
- (3) a host cell (IV) comprising (III);

(4) production of (I);
 (5) a transgenic non-human animal that expresses a human (I);
 (6) screening (M) for a modulator of Smurf activity, comprising detecting modulation of Smurf activity in the presence of a test compound relative to Smurf activity in the absence of the test compound;
 (7) an antibody (V) that specifically binds to (I);
 (8) an oligonucleotide or nucleic acid (VI) that specifically hybridizes to (II) under highly stringent conditions; and
 (9) promoting a bone morphogenic protein or transforming growth factor (TGF)- beta activation pathway in a cell, comprising suppressing expression of endogenous Smurf in the cell.

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - Negative regulator of Smad signal transduction; antagonist of BMP and TGF- beta signaling pathway.

The inhibition of Smad1 by Smurf1 was tested. By over expressing Smad1 and Smad2 together with various dosages of Smurf1 in Xenopus animal caps, the ability of Smurf1 to directly antagonize the mesoderm induction activities of Smad1 and Smad2, was tested. The results showed that expression of Smad1 alone induced ventral mesoderm, as demonstrated by expression of the ventral/posterior mesodermal markers Xhox3 and Xcad1. However, co-expression of Smurf1 and Smad1 blocked induction of these markers at all Smurf1 doses tested, demonstrating that Smurf1 can antagonize Smad1 activity.

USE - Expression of (I) from (III) in a cell is useful for inhibiting a bone morphogenic protein (BMP) or transforming growth factor- beta (TGF beta) activation pathway in a cell (claimed). (I) is useful to block chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. (I) is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it by antagonizing or mimicking the activity of (I), respectively, and in screening assays for identifying specific ligands of (I). (I) is useful as an immunogen to generate antibodies that are useful to alter the BMP pathway by inhibiting (I) or for diagnostic purposes. (I) is useful for treating a disorder associated with BMP or TGF- beta activation, such as cancer. (I) or inhibitor of (I) can be delivered by a vector to modulate Smads, e.g. to prevent Smurf regulation of Smads where BMP or TGF beta activity is desired, such as in bone regeneration or to study Smurf regulator processes in vivo.

Dwg.0/18

ACCESSION NUMBER: 2001-071267 [08] WPIDS
 DOC. NO. CPI: C2001-019969
 TITLE: Novel isolated Smurf protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.
 DERWENT CLASS: B04 D16
 INVENTOR(S): THOMSEN, G H; WRANA, J
 PATENT ASSIGNEE(S): (HSCR-N) HSC RES & DEV LP; (UYN) UNIV NEW YORK STATE RES FOUND
 COUNTRY COUNT: 93
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000077168	A2	20001221	(200108)*	EN	106
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000056107	A	20010102	(200121)		
EP 1192174	A2	20020403	(200230)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT					

RO SE SI
JP 2003502064 W 20030121 (200308) 131
CN 1409722 A 20030409 (200345)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000077168	A2	WO 2000-US16250	20000612
AU 2000056107	A	AU 2000-56107	20000612
EP 1192174	A2	EP 2000-941398	20000612
		WO 2000-US16250	20000612
JP 2003502064	W	WO 2000-US16250	20000612
		JP 2001-504003	20000612
CN 1409722	A	CN 2000-811354	20000612

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000056107	A Based on	WO 2000077168
EP 1192174	A2 Based on	WO 2000077168
JP 2003502064	W Based on	WO 2000077168

PRIORITY APPLN. INFO: US 1999-138969P 19990611

L1 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI ENaC degradation in A6 cells by the ubiquitin-proteosome proteolytic
pathway.
AB Amiloride-sensitive epithelial Na⁺ channels (ENaC) are responsible for
trans-epithelial Na⁺ transport in the kidney, lung, and colon. The
channel consists of three subunits (alpha, beta, gamma) each containing a
proline rich region (PPXY) in their carboxyl-terminal end. Mutations in
this **PPXY domain** cause Liddle's syndrome, an autosomal
dominant, salt-sensitive hypertension, by preventing the channel's
interactions with the ubiquitin ligase Neural precursor cell-expressed
developmentally down-regulated protein (Nedd4). It is postulated that
this results in defective endocytosis and lysosomal degradation of ENaC
leading to an increase in ENaC activity. To show the pathway that
degrades ENaC in epithelial cells that express functioning ENaC channels,
we used inhibitors of the proteosome and measured sodium channel activity.
We found that the inhibitor, MG-132, increases amiloride-sensitive
trans-epithelial current in Xenopus distal nephron A6 cells. There also
is an increase of total cellular as well as membrane-associated ENaC
subunit molecules by Western blotting. MG-132-treated cells also have
increased channel density in patch clamp experiments. Inhibitors of
lysosomal function did not reproduce these findings. Our results suggest
that in native renal cells the proteosomal pathway is an important
regulator of ENaC function.

ACCESSION NUMBER: 2001:301269 BIOSIS
DOCUMENT NUMBER: PREV200100301269
TITLE: ENaC degradation in A6 cells by the ubiquitin-proteosome
proteolytic pathway.
AUTHOR(S): Malik, Bela [Reprint author]; Schlanger, Lynn; Al-Khalili,
Otor; Bao, Hui-Fang; Yue, Guichun; Price, Stephen Russ;
Mitch, William E.; Eaton, Douglas Charles
CORPORATE SOURCE: Dept. of Physiology, Center for Cell and Molecular
Signaling, 1648 Pierce Dr., Physiology Bldg., Rm. 074,
Atlanta, GA, 30322, USA
bmalik@ccms-renal.physio.emory.edu
SOURCE: Journal of Biological Chemistry, (April 20, 2001) Vol. 276,
No. 16, pp. 12903-12910. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Jun 2001
Last Updated on STN: 19 Feb 2002

=> s Smad polypeptide
L2 65 SMAD POLYPEPTIDE

=> s l2 and Smurf activity
L3 0 L2 AND SMURF ACTIVITY

=> s smurf activity
L4 1 SMURF ACTIVITY

=> d l4 ti abs ibib tot

L4 ANSWER 1 OF 1 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Novel isolated Smurf protein useful for inhibiting bone morphogenic
protein or tumor growth factor-beta activation pathway, for treating
cancer and to block osteogenesis, hair growth, tooth formation.

AN 2001-071267 [08] WPIDS
AB WO 200077168 A UPAB: 20011129

NOVELTY - An isolated Smurf1 or Smurf2 protein (I), is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the
following:

- (1) an isolated nucleic acid (II) encoding (I);
- (2) a vector (III) comprising (II);
- (3) a host cell (IV) comprising (III);
- (4) production of (I);
- (5) a transgenic non-human animal that expresses a human (I);
- (6) screening (M) for a modulator of **Smurf activity**
, comprising detecting modulation of **Smurf activity** in
the presence of a test compound relative to **Smurf**
activity in the absence of the test compound;
- (7) an antibody (V) that specifically binds to (I);
- (8) an oligonucleotide or nucleic acid (VI) that specifically
hybridizes to (II) under highly stringent conditions; and
- (9) promoting a bone morphogenic protein or transforming growth
factor (TGF)- beta activation pathway in a cell, comprising suppressing
expression of endogenous Smurf in the cell.

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - Negative regulator of Smad signal transduction;
antagonist of BMP and TGF- beta signaling pathway.

The inhibition of Smad1 by Smurf1 was tested. By over expressing
Smad1 and Smad2 together with various dosages of Smurf1 in Xenopus animal
caps, the ability of Smurf1 to directly antagonize the mesoderm induction
activities of Smad1 and Smad2, was tested. The results showed that
expression of Smad1 alone induced ventral mesoderm, as demonstrated by
expression of the ventral/posterior mesodermal markers Xhox3 and Xcad1.
However, co-expression of Smurf1 and Smad1 blocked induction of these
markers at all Smurf1 doses tested, demonstrating that Smurf1 can
antagonize Smad1 activity.

USE - Expression of (I) from (III) in a cell is useful for inhibiting
a bone morphogenic protein (BMP) or transforming growth factor- beta (TGF
beta) activation pathway in a cell (claimed). (I) is useful to block
chondrogenesis, osteogenesis, blood differentiation, cartilage formation,
neural tube patterning, retinal development, heart induction and
morphogenesis, hair growth, tooth formation, gamete formation and a wide
variety of tissue and organ formation processes, and hinder the
regeneration, growth, maintenance, etc., of bone and other tissues that
are dependent on the BMP pathway. (I) is useful for screening for various
drugs and/or antibodies that can either enhance the BMP pathway, or
inhibit it by antagonizing or mimicking the activity of (I), respectively,
and in screening assays for identifying specific ligands of (I). (I) is
useful as an immunogen to generate antibodies that are useful to alter the
BMP pathway by inhibiting (I) or for diagnostic purposes. (I) is useful
for treating a disorder associated with BMP or TGF- beta activation, such
as cancer. (I) or inhibitor of (I) can be delivered by a vector to
modulate Smads, e.g. to prevent Smurf regulation of Smads where BMP or TGF
beta activity is desired, such as in bone regeneration or to study Smurf
regulator processes in vivo.

Dwg.0/18

ACCESSION NUMBER: 2001-071267 [08] WPIDS
 DOC. NO. CPI: C2001-019969
 TITLE: Novel isolated Smurf protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.
 DERWENT CLASS: B04 D16
 INVENTOR(S): THOMSEN, G H; WRANA, J
 PATENT ASSIGNEE(S): (HSCR-N) HSC RES & DEV LP; (UYNY) UNIV NEW YORK STATE RES FOUND
 COUNTRY COUNT: 93
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000077168	A2	20001221	(200108)*	EN	106
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000056107	A	20010102	(200121)		
EP 1192174	A2	20020403	(200230)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2003502064	W	20030121	(200308)		131
CN 1409722	A	20030409	(200345)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000077168	A2	WO 2000-US16250	20000612
AU 2000056107	A	AU 2000-56107	20000612
EP 1192174	A2	EP 2000-941398	20000612
		WO 2000-US16250	20000612
JP 2003502064	W	WO 2000-US16250	20000612
		JP 2001-504003	20000612
CN 1409722	A	CN 2000-811354	20000612

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000056107	A Based on	WO 2000077168
EP 1192174	A2 Based on	WO 2000077168
JP 2003502064	W Based on	WO 2000077168

PRIORITY APPLN. INFO: US 1999-138969P 19990611

=> e thomsen,g/au

E1	2	THOMSEN ZIEGER N/AU
E2	4	THOMSEN ZIEGER NADINE/AU
E3	0 -->	THOMSEN,G/AU
E4	1	THOMSENE T/AU
E5	1	THOMSENK/AU
E6	1	THOMSENS P/AU
E7	12	THOMSER J/AU
E8	1	THOMSERN J B/AU
E9	2	THOMSETT A/AU
E10	1	THOMSETT C E/AU
E11	1	THOMSETT D W/AU
E12	1	THOMSETT E C/AU

=> e wrana,j/au

E1	2	WRANA M/AU
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E2	1	WRANA MICHAEL/AU
E3	0 -->	WRANA, J/AU
E4	1	WRANAWIN K/AU
E5	1	WRANCE O/AU
E6	1	WRANCKEN A/AU
E7	1	WRANEK J/AU
E8	1	WRANEK P/AU
E9	41	WRANEK U/AU
E10	3	WRANEK URSULA/AU
E11	2	WRANELL L/AU
E12	3	WRANES E/AU